PAPERS AND SHORT REPORTS

Epileptic dizziness

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Abstract

Clinical and electroencephalographic features and the response to treatment of 30 patients with episodic dizziness due to epilepsy were noted. The symptom consisted of a brief episode of disequilibrium, often with a sensation of rotation, without evident precipitating factors or sequelae.

A history of "absences" or other features suggestive of temporal lobe epilepsy was elicited in over half the patients, and seven (almost a quarter) had had one or more generalised seizures before presentation. Electroencephalography showed a posterior temporal lobe focus in all but two patients, and there was a family history of epilepsy in six. Response to treatment with phenytoin or carbamazepine was good in most patients.

Epilepsy should be considered in the differential diagnosis of episodic dizziness or vertigo, especially in young people.

Introduction

Although dizziness as a manifestation of epilepsy was recognised 100 years ago by Hughlings Jackson[1] and later by Gowers,[2] the possibility that brief episodes of dizziness may be due to epilepsy[3] is not well recognised. In our experience epilepsy is an important cause of transient dizziness, with or without a rotational component. We describe the clinical and electroencephalographic features and the results of treatment in 30 adults whose presenting complaint of episodic dizziness was found to be due to epilepsy.

Patients, methods, and results

We studied 30 consecutive patients aged 15-65 (mean 35) years. Eighteen were women. Dizziness, defined as a transient sense of disequilibrium, with or without a feeling of rotation, was the sole or main reason for referral. In all patients investigation and the results of treatment indicated that the dizziness was due to epilepsy rather than any other disorder. Twenty-four patients were referred direct by their general practitioners, three from the ear, nose, and throat clinic at this hospital, and three by other doctors. Patients in whom dizziness was not the predominant symptom were excluded, as were those older than 65 years, those with a history of cerebral vascular disease, and those with symptoms of middle-ear disease.

Each patient's assessment was based on the history and examination results, but all underwent otological and electroencephalographic assessments. The otological assessment consisted of clinical, audiometric, and caloric tests. Radiological investigations, including radioisotopic scanning or computed tomography of the head, were carried out in five patients. The electroencephalographic assessment consisted of routine waking records supplemented by recordings made during hyperventilation and intermittent photic stimulation. Sleep recordings were made in four patients.

When the diagnosis of epileptic dizziness had been established treatment was started with anticonvulsant drugs. The period of follow-up varied from three months to eight years (mean 2·7 years). The assessments of six patients, who had moved away from east London, were based on their replies to a questionnaire, but the remaining patients were assessed in the clinic.

SYMPTOMS

In all 30 patients dizziness occurred in brief episodes, each lasting no more than a few seconds. A feeling of rotation was described by 14 patients (47%). The symptom usually began abruptly as dizziness or as a feeling of instability that evolved to dizziness or rotational vertigo during several seconds. In the recovery phase nausea was often experienced. Attacks were not related to postural change or to any other definite external factor. The frequency of the attacks varied between patients, and in individual patients at different times, ranging from about one a week to several episodes daily. Symptomatic dizziness had been present for from six months to 42 years (mean 10 years), but in almost two-thirds (19) of the patients it had occurred only during the previous one to five years. The mean age of onset was 25 years.

By the time of their first attendance seven patients (23%) had also experienced generalised convulsions. In six of these patients episodic dizziness had preceded the development of generalised seizures by from six weeks to 17 months; in one patient the illness began with a major seizure, itself preceded by a dizzy feeling, followed by frequent episodes of isolated vertigo. In the seven patients with generalised seizures dizzy spells occurred both separately and immediately preceding generalised seizures.

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Other symptoms were also noted and usually occurred separately from the episodes of dizziness. The commonest was brief "absences" in 15 patients, in which short periods of altered consciousness occurred, without loss of posture. Sometimes the patient would retain awareness of the environment but be momentarily unable to move or speak. Other symptoms were less common and included generalised convulsions (seven patients), depersonalisation (seven), epigastric discomfort (six), nausea (six), headaches (five), a sense of déjà vu (two), anxiety or panic (two), automatisms (two), auditory hallucinations (two), and gustatory hallucinations (one).

Only one patient showed evidence of an acquired cerebral lesion; this patient had sustained an injury to the head 13 years earlier. Two patients (7%) had experienced febrile convulsions in infancy, and six others (20%) had a family history of epilepsy. In three of these families dizziness was recognised as a manifestation of epilepsy in affected relatives.

SIGNS

Results of clinical examination and tests of hearing and vestibular function were normal in all patients. Radiological investigation results were also normal.

ELECTROENCEPHALOGRAPHY

An abnormal electroencephalogram was a major criterion for diagnosis. In all but two patients the abnormality consisted of temporal or bitemporal sharp or slow wave foci. In some cases there were associated generalised seizure discharges. This electroencephalographic disturbance had a left posterior temporal emphasis in 15 patients, a right temporal emphasis in seven, and a bitemporal pattern in six. Electroencephalography in the remaining two patients showed a generalised atypical spike and wave abnormality and a diffuse paroxysmal theta pattern respectively. Both these patients had a family history of epilepsy. In four patients the electroencephalographic disturbance was dubious in routine recordings but confirmed in subsequent sleep recordings.

TREATMENT

Several different diagnoses had been considered before the patients had been referred for neurological opinion, including Menière's disease, cervical spondylosis, middle-ear disease, cerebral vascular disease, and anxiety and depression, and most patients had therefore tried various drugs. These often included phenothiazines, benzodiazepines, and tricyclic antidepressants, which were usually ineffective. Two patients in whom "absences" had been frequent had been treated with tridione and ethosuccimide respectively without effect on their dizziness.

Treatment with phenytoin or carbamazepine was effective. In the 23 patients (77%) in whom dizziness occurred without generalised seizures there was complete remission in 10 (mean follow-up 18 months) and a considerable reduction in the frequency and severity of attacks in the remainder (mean follow-up two years). Six of the seven patients who had also had generalised seizures were completely free of both dizziness and other manifestations of epilepsy during one and a half to five years of follow-up. The patient with persisting seizures and dizzy episodes did not comply with treatment.

Discussion

Dizziness is a common symptom but diagnosis is not always easy. It may result from dysfunction of the vestibular system at any point from the ear to the cerebral cortex. Dizziness due to labyrinthine disease or brain-stem lesions, especially cerebral vascular disease, is particularly common and usually recognised by its typical associated features. Although in these instances dizziness may occasionally occur in isolation, the associated clinical features are usually diagnostic. Furthermore, in these disorders the onset occurs later than in our patients, whose mean age was only 25 years. Dizziness may also occur in patients with multiple sclerosis, but this disorder may be recognised by its associated clinical features. Hyperventilation associated with anxiety or emotional distress may cause dizziness, with or without tetany, but in such patients electroencephalography shows no features of epilepsy. Epileptic dizziness may often appear as part of an aura in generalised seizures but is then usually poorly defined. It is more specifically related, however, to temporal lobe epilepsy, occurring as a component of temporal lobe attacks in 126 (19%) out of 666 patients studied by Currie *et al.*[3] Our patients differ from theirs since, although the epileptic dizziness was associated with clinical and electroencephalographic evidence of temporal lobe epilepsy, it occurred as a repeated, isolated, episodic symptom; this is a less common occurrence. The 30 patients described here presented during a two-year period, representing an incidence of less than 1% of all new cases of epilepsy.

Hughlings Jackson[1] recognised that in temporal lobe epilepsy dizziness is not simply an aura but usually constitutes part of the seizure. Indeed, dizziness may be its sole manifestation. This was indicated in our patients by the paroxysmal quality of the symptom, its close temporal relation with a generalised convulsion in some patients, and the associated electroencephalographic abnormality. The dizziness itself was often characteristic, consisting of sudden very brief episodes, lasting only a few seconds, followed by rapid recovery without sequelae. These episodes usually occurred without associated symptoms of epilepsy. Contrary to common belief that a subjective sensation of rotation is almost exclusively a manifestation of labyrinthine disease, rotation was experienced by nearly half our patients in their attacks. The diagnosis of epileptic dizziness was often suspected because of the occurrence of separate symptoms of temporal lobe epilepsy, often elicited only by careful questioning, and was confirmed by finding typical electroencephalographic abnormalities and by the response to treatment with appropriate anticonvulsant drugs.

The vestibular system projects to the posterior part of the superior temporal and middle temporal convolutions of the temporal lobes and thence to the supramarginal and angular gyri.[4,5] The relatively posterior location of the vestibular representation in the temporal lobes probably accounts for the isolation of the symptom of dizziness or vertigo from other symptoms of temporal lobe epilepsy in our patients.[3] Previous reports have distinguished vestibular and vestibulogenic seizures.[6] In vestibular seizures the dizziness originates, as in our patients, from abnormal discharges in the cortical vestibular centres, while in vestibulogenic seizures the attack is said to be initiated by labyrinthine discharges, often from an abnormal labyrinth, which excite epileptic activity in neurones in the brain stem and temporal lobe.[6] There was no evidence to support the suggestion of vestibulogenic seizures in our patients. There have been few other reports of epileptic dizziness.[7-9] Drachman and Hart[7] found no instance in 125 patients investigated for dizziness, but Hughes and Drachman[8] found that electroencephalographic abnormalities were more common in dizzy patients and that dizziness was more common in epileptic patients than controls. Pedersen and Jepsen[9] recognised the concept but did not require electroencephalographic abnormality for diagnosis. In more than half of a group of 50 children with dizziness unassociated with evidence of middle-ear or brain-stem disease, however, Evistar and Evistar[10] found that epilepsy was the underlying cause.

A family history of epilepsy of similar character was elicited in three (10%) of our patients. Although this suggests the possibility of a specific genetic element, the numbers are too small to allow firm conclusions to be drawn.

The underlying cause of the temporal lobe epilepsy was uncertain in many cases. In some the electroencephalographic abnormality suggested a diagnosis of idiopathic epilepsy. There was evidence of an acquired cause in one patient, who had a history of a previous head injury. Radiological investigation was deferred in all but five patients because the electroencephalographic abnormality did not suggest a focal progressive lesion.

In most of our patients several other diagnoses had been considered for some months before a neurological opinion was sought despite the absence of other clinical features, such as deafness, brain-stem symptoms, or affective change, to support

these diagnoses. No doubt the transitory nature of the dizziness, and the patients' perfect health between attacks of dizziness must have contributed to the tendency to consider the attacks psychogenic. Despite the length of the history of dizzy episodes in many of the patients and the development of generalised seizures in seven, anticonvulsant medication was extremely effective. It is thus clearly of great practical importance to consider epilepsy in the differential diagnosis of episodic dizziness or vertigo, especially in young people.

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The Cardiff Cervical Cytology Study: prevalence of cytological grades and initial histological findings

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Abstract

Among 45 266 women in the Cardiff Cervical Cytology Survey the peak prevalence of suspicious or positive smears was 11·2/1000 at age 45-50 years and of dyskaryosis 10·2/1000 at age 25-29. A suspicious or positive cytological picture at prevalence testing was associated with occult or clinical invasion in 24% of cases, and only 4% of patients with suspicious or positive smears were normal histologically. When dyskaryosis was detected in the prevalence test 20% had carcinoma in situ or microinvasion and 3% had occult or clinically invasive carcinoma. One hundred and twenty-nine (51%) women with dyskaryotic smears did not have a biopsy initially (that is, within two years of the prevalence test), but they were followed up at regular intervals. Subsequently 15 of the 129 gave smears consistently dyskaryotic or worse cytologically and were subjected to biopsy. Of these, two showed dysplasia, 12 carcinoma in situ, and one clinically invasive carcinoma.

These findings emphasise the need for repeat cytological or histological examination in any woman with evidence of dyskaryosis in a cervical smear.

Introduction

The benefits of screening programmes for cervical neoplasia are still not fully evaluated. The optimum strategy, particularly in terms of frequency of screening and the age at which screening should first be done, is uncertain and will remain so until more is known about the natural history of cancer.

This paper, based on data from the Cardiff Cervical Cytology Study,[1 2] describes the prevalence of the various cytological patterns and the histological findings within two years of the prevalence test.

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Subjects and methods

The study was based on a defined population of all "ever-married" women aged 25-69 resident within the Cardiff City area. Methods, enumeration and definition of the population, and initial acceptance rates have been described.[1 2] The total defined population was 70 869 women, of whom 45 915 (65%) had had at least one cervical smear test before the end of February 1971, when entry to the study was terminated.[2] This analysis is based on 45 266 women of known age who had not had hysterectomy, treated invasive cancer of the cervix, or other gynaecological cancer.

CYTOLOGY

Smears were taken with a wooden spatula and stained by the Papanicolaou technique. The quality of the smears was assessed as good, poor, or useless. In the latter case repeat samples were requested. Both good and poor quality smears were classified on a five-point scale as: normal (none of the following); atypical (changes of the order seen in inflammation); dyskaryosis (irregularities in size, shape, and chromatin pattern of nucleus—subdivided as mild, moderate, or severe); suspicious (changes considered suspicious of malignancy); and positive (changes characteristic of malignancy).

PREVALENCE TEST AND RESULT

The *prevalence test* was defined as the first cervical smear test performed between 1965 (when the study began) and February 1971 (when entry to the study closed). For 96% of women this was their first known smear. About 4000 had at least one repeat test within three months of their prevalence test, either as part of a reproducibility study or to confirm an abnormal smear. To take account of these repeat smears the cytological *prevalence result* was defined as the worst cytological appearances at or within three months of the prevalence test.

CLINICAL MANAGEMENT

The recall system and initial management have been described[1] but are summarised here. Any woman in whom a gynaecological abnormality was detected (whatever the smear result) was referred either to her general practitioner—for example, for minor infections—or, if there was any suspicion of gynaecological malignancy, direct to the gynaecologist. In the absence of clinically evident pelvic disease the following recall programme was used.

Normal cytological appearance—Repeat smear after two to three years.